Search Results -

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l4 and l5	15

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Refine Search: `l4 and l5` Clear

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DB Name	Query	Hit Count	Set Name
USPT,PGPB	l4 and l5	15	L6
USPT,PGPB	(myocyte or muscle) adj specific adj promoter	60	L5
USPT,PGPB	l3 and (cmv near5 promoter)	519	L4
USPT,PGPB	l1 and l2	1876	L3
USPT,PGPB	(eia and e1b) or e1	25286	L2
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☐ 1. Document ID: US 6331527 B1

L6: Entry 1 of 15 File: USPT Dec 18, 2001

US-PAT-NO: 6331527
DOCUMENT-IDENTIFIER: US 6331527 B1

TITLE: Promoter smooth muscle cell expression

DATE-ISSUED: December 18, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Parmacek; Michael S.	Bryn Mawr	PA		
Solway; Julian	Glencoe	IL		

US-CL-CURRENT: 514/44; 435/455, 435/456, 536/24.1, 623/1.13, 623/1.41

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 6306830 B1

L6: Entry 2 of 15 File: USPT Oct 23, 2001

US-PAT-NO: 6306830
DOCUMENT-IDENTIFIER: US 6306830 B1

TITLE: Gene therapy for congestive heart failure

DATE-ISSUED: October 23, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hammond; H. Kirk	La Jolla	CA		
Insel; Paul A.	San Diego	CA		
Ping; Peipei	Jeffersonville	IN		
Post; Steven R.	Lexington	KY		
Gao; Meihua	San Diego	CA		

US-CL-CURRENT: 514/44; 424/93.2, 435/235.1, 435/320.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 6297221 B1

L6: Entry 3 of 15 File: USPT Oct 2, 2001

US-PAT-NO: 6297221
DOCUMENT-IDENTIFIER: US 6297221 B1

TITLE: Method for promoting angiogenesis with a nucleic acid construct comprising an SM22.alpha.0 promoter

DATE-ISSUED: October 2, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Parmacek; Michael S.	Chicago	IL		
Solway; Julian	Glencoe	IL		

US-CL-CURRENT: 514/44

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

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☐ 4. Document ID: US 6291211 B1

L6: Entry 4 of 15 File: USPT Sep 18, 2001

US-PAT-NO: 6291211
DOCUMENT-IDENTIFIER: US 6291211 B1

TITLE: Promoter for smooth muscle cell expression

DATE-ISSUED: September 18, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Parmacek; Michael S.	Chicago	IL		
Solway; Julian	Glencoe	IL		

US-CL-CURRENT: 435/69.1; 435/455, 435/456, 435/91.3, 514/44

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 6290949 B1

L6: Entry 5 of 15 File: USPT Sep 18, 2001

US-PAT-NO: 6290949
DOCUMENT-IDENTIFIER: US 6290949 B1

TITLE: Adenoviral vector for inhibiting restenosis

DATE-ISSUED: September 18, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
French; Brent A.	Houston	TX	77096	
Raizner; Albert E.	Houston	TX	77024	
Roberts; Robert	Houston	TX	77019	

US-CL-CURRENT: 424/93.2; 424/93.6, 435/320.1, 435/456, 514/44

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 6. Document ID: US 6284743 B1

L6: Entry 6 of 15 File: USPT Sep 4, 2001

US-PAT-NO: 6284743
DOCUMENT-IDENTIFIER: US 6284743 B1

TITLE: Method for modulating smooth muscle cell proliferation

DATE-ISSUED: September 4, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Parmacek; Michael S.	Chicago	IL		
Solway; Julian	Glencoe	IL		

US-CL-CURRENT: 514/44; 435/375, 435/69.1

Full | Title | Citation | Front | Review | Classification | Date | Reference KWMC | Draw Desc | Image

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☐ 7. Document ID: US 6271211 B1

L6: Entry 7 of 15 File: USPT Aug 7, 2001

US-PAT-NO: 6271211
DOCUMENT-IDENTIFIER: US 6271211 B1

TITLE: Gene therapy for regulating penile smooth muscle tone

DATE-ISSUED: August 7, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Christ; George J.	Smithtown	NY		
Melman; Arnold	Ardsley	NY		

US-CL-CURRENT: 514/44; 435/320.1, 435/325, 435/455, 530/350, 536/23.1, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference KWMC | Draw Desc | Image

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☐ 8. Document ID: US 6218179 B1

L6: Entry 8 of 15 File: USPT Apr 17, 2001

US-PAT-NO: 6218179
DOCUMENT-IDENTIFIER: US 6218179 B1

TITLE: Tissue specific hypoxia regulated constructs

DATE-ISSUED: April 17, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Webster; Keith A.	Key Biscayne	CA		
Bishopric; Nanette H.	Key Biscayne	CA		
Murphy; Brian	Palo Alto	CA		
Laderoute; Keith R.	Menlo Park	CA		
Green; Christopher J.	Novato	CA		

US-CL-CURRENT: 435/320.1; 435/325, 435/455, 536/23.1, 536/24.1

☐ 9. Document ID: US 6174871 B1

L6: Entry 9 of 15 File: USPT Jan 16, 2001

US-PAT-NO: 6174871
DOCUMENT-IDENTIFIER: US 6174871 B1

TITLE: Gene therapies for enhancing cardiac function

DATE-ISSUED: January 16, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hammond; H. Kirk	La Jolla	CA		
Giordano; Frank J.	Del Mar	CA		
Dillmann; Wolfgang H.	Solana Beach	CA		

US-CL-CURRENT: 514/44; 424/93.6, 435/320.1, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 10. Document ID: US 6114311 A

L6: Entry 10 of 15 File: USPT Sep 5, 2000

US-PAT-NO: 6114311
DOCUMENT-IDENTIFIER: US 6114311 A

TITLE: Method for modulating smooth muscle cell proliferation

DATE-ISSUED: September 5, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Parmacek; Michael S.	Chicago	IL		
Solway; Julian	Glencoe	IL		

US-CL-CURRENT: 514/44

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 11. Document ID: US 6090618 A

L6: Entry 11 of 15 File: USPT Jul 18, 2000

US-PAT-NO: 6090618
DOCUMENT-IDENTIFIER: US 60906 A

TITLE: DNA constructs and viral vectors comprising a smooth muscle promoter

DATE-ISSUED: July 18, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Parmacek; Michael S.	Chicago	IL		
Solway; Julian	Glencoe	IL		

US-CL-CURRENT: 435/320.1; 536/23.1, 536/23.5, 536/24.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 12. Document ID: US 6017734 A

L6: Entry 12 of 15 File: USPT Jan 25, 2000

US-PAT-NO: 6017734
DOCUMENT-IDENTIFIER: US 6017734 A

TITLE: Unique nucleotide and amino acid sequence and uses thereof

DATE-ISSUED: January 25, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Summers; Max D.	Bryan	TX		
Braunagel; Sharon C.	Bryan	TX		
Hong; Tao	Bryan	TX		

US-CL-CURRENT: 435/69.7; 435/320.1, 435/348, 435/365, 435/91.4, 536/23.1, 536/23.72, 536/24.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 13. Document ID: US 5994104 A

L6: Entry 13 of 15 File: USPT Nov 30, 1999

US-PAT-NO: 5994104
DOCUMENT-IDENTIFIER: US 5994104 A

TITLE: Interleukin-12 fusion protein

DATE-ISSUED: November 30, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Anderson; Robert James	London			GBX
Prentice; Hugh Grant	London			GBX
MacDonald; Ian Duncan	London			GBX

US-CL-CURRENT: 435/69.52; 424/85.2, 435/252.3, 435/320.1, 435/325, 435/69.5, 435/69.51, 435/69.7, 530/351, 536/23.4, 536/23.5, 930/141

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 14. Document ID: US 5885829 A

L6: Entry 14 of 15 File: USPT Mar 23, 1999

US-PAT-NO: 5885829
DOCUMENT-IDENTIFIER: US 5885829 A

TITLE: Engineering oral tissues

DATE-ISSUED: March 23, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mooney; David J.	Ann Arbor	MI		
Rutherford; Robert B.	Ann Arbor	MI		

US-CL-CURRENT: 435/325; 424/422, 424/435, 424/49, 435/374, 435/378, 435/69.1

Full | Title | Citation | Front | Review | Classification | Date | Reference — KWIC | Draw Desc | Image

☑ 15. Document ID: US 5658729 A

L6: Entry 15 of 15 File: USPT Aug 19, 1997

US-PAT-NO: 5658729
DOCUMENT-IDENTIFIER: US 5658729 A

TITLE: Method, reagent and kit for evaluating susceptibility to premature atherosclerosis

DATE-ISSUED: August 19, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hayden; Michael R.	Vancouver			CAX
Ma; Yuanhong	Los Altos	CA		
Lewis; Suzanne	West Vancouver			CAX
Liu; Guoquing	Vancouver			CAX

US-CL-CURRENT: 435/6; 435/810, 435/91.1, 435/91.2, 536/23.2, 536/24.3, 536/24.31, 536/24.33, 536/24.5

Full | Title | Citation | Front | Review | Classification | Date | Reference — KWIC | Draw Desc | Image

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(FILE 'HOME' ENTERED AT 17:14:40 ON 25 JAN 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 17:14:54 ON 25 JAN 2002

L1 2379 S HSP72
L2 2474033 S DNA OR CDNA OR POLYNUCLEOTIDE
L3 296 S L1 AND L2
L4 92 S (DNA OR CDNA OR POLYNUCLEOTIDE)(8A)HSP72
L5 31 DUP REM L4 (61 DUPLICATES REMOVED)
L6 40 S HSP72(3A)(DNA OR CDNA)
L7 16 DUP REM L6 (24 DUPLICATES REMOVED)
L8 196 S HSP72(5A)GENE
L9 124 S HSP72(W)GENE
L10 42 DUP REM L9 (82 DUPLICATES REMOVED)

=> d 30-42 au ti so ab l10

L10 ANSWER 30 OF 42 MEDLINE DUPLICATE 21
AU Fink S L; Chang L K; Ho D Y; Sapolsky R M
TI Defective herpes simplex virus vectors expressing the rat brain stress-inducible heat shock protein 72 protect cultured neurons from severe heat shock.
SO JOURNAL OF NEUROCHEMISTRY, (1997 Mar) 68 (3) 961-9.
Journal code: JAV; 2985190R. ISSN: 0022-3042.
AB Recently, preinduction of the heat shock response has been shown to protect CNS neurons undergoing various stressful insults, e.g., heat, ischemia, or exposure to excitotoxins. However, it is not known which of the proteins induced by the heat shock response mediate the protective effects. Previous correlative evidence points to a role for the highly stress-induced 72-kDa heat shock protein (hsp72). However, it is not known whether hsp72 expression alone can protect against a range of acute neuronal insults. We constructed a herpes simplex virus-1 vector carrying the rat brain stress-inducible **hsp72 gene** and the Escherichia coli lacZ (marker) gene. Infection with the vector caused hippocampal neurons to coexpress hsp72 and beta-galactosidase. Infection with a control vector led to marker gene expression only. Overexpression of hsp72 protected cultured hippocampal neurons against a heat shock but not against the metabolic toxin 3-nitropropionic acid or the excitotoxin glutamate. This is the first published report of protection following heat shock protein transfection in CNS neurons.

L10 ANSWER 31 OF 42 MEDLINE DUPLICATE 22
AU Zhou M; Wu X; Ginsberg H N
TI Evidence that a rapidly turning over protein, normally degraded by proteasomes, regulates **hsp72 gene** transcription in HepG2 cells.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 Oct 4) 271 (40) 24769-75.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
AB Heat shock protein 72/73 (Hsp70) is a cytosolic molecular chaperone that carries out fundamental roles under both normal and stress situations. There is great interest in delineating the mechanisms whereby Hsp70 levels are regulated. We observed that N-acetyl-leucyl-leucyl-norleucinal (ALLN), a synthetic aldehydic tripeptide that inhibits proteasomes, markedly

induced Hsp70 levels (up to 30-fold above base line in HepG2 cells and human endothelial cells). Induction of Hsp70 by ALLN was dose-dependent and not related to cell toxicity. ALLN selectively increased Hsp70 levels without affecting Hsp25, Hsp27, Hsp60, Hsp86, Hsp90, Hsp104, or Bip (immunoglobulin heavy chain binding protein) in HepG2 cells. ALLN induced Hsp70 not only by stabilizing the protein but also by dramatically increasing its synthesis. The modulation of Hsp70 synthesis by ALLN resulted from a rapid and marked increase in transcription of the **hsp72 gene**, since the induction of hsp72 mRNA was blocked in cells co-treated with actinomycin D. hsp72 mRNA levels were affected in a time-dependent manner by exposure to ALLN; significant elevations occurred within 60 min of treatment, and a decline to background levels was observed by 7 h of recovery. The ALLN-induced increase in **hsp72 gene** expression was associated with trimerization of the heat shock transcriptional factor (HSF1). ALLN did not affect the steady-state level of HSF1 protein. The effects of ALLN appeared to require de novo protein synthesis, since the induction of both HSF1 trimerization and hsp72 transcription was blocked by co-treatment with cycloheximide. When we tested a series of protease inhibitors, only the related aldehydic tripeptides, N-acetyl-leucyl-leucyl-methioninal and the proteasome inhibitor, Cbz-leucyl-leucyl-leucinal, induced Hsp70 levels. The specific proteasome inhibitor, lactacystin, which has a different structure, also induced Hsp70 levels. Overall, our results suggest that a rapidly turning over protein that is normally degraded by proteasomes may be involved in the regulation of Hsp70 synthesis via effects on the hsp70 transcriptional factor, HSF1.

L10 ANSWER 32 OF 42 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 23
AU Madonna, M. B. (1); Nelson, M. S.; Busch, M. W.; Chang, E. B.
TI Retroviral delivery of human heat shock protein 72 (**hsp72**) **gene** into intestinal IEC-18 cells.
SO Gastroenterology, (1996) Vol. 110, No. 4 SUPPL., pp. A953.
Meeting Info.: 96th Annual Meeting of the American Gastroenterological Association and the Digestive Disease Week San Francisco, California, USA May 19-22, 1996
ISSN: 0016-5085.

L10 ANSWER 33 OF 42 MEDLINE DUPLICATE 24
AU Ohnishi K; Matsumoto H; Takahashi A; Wang X; Ohnishi T
TI Heat shock transcription factor, HSF, is activated by ultraviolet irradiation.
SO PHOTOCHEMISTRY AND PHOTOBIOLOGY, (1996 Dec) 64 (6) 949-52.
Journal code: P69; 0376425. ISSN: 0031-8655.
AB We demonstrated previously that human glioblastoma cell lines accumulated heat shock protein (hsp)72, not only after heat shock, but also after, gamma-ray or UV irradiation. In the present study, we investigated whether the binding activity of heat shock transcription factor (HSF) to the heat shock element (HSE) of the **hsp72 gene** promoter increased after UV irradiation of human glioblastoma A-172 cells. A gel mobility-shift assay showed that the activated HSF level increased markedly after UV irradiation. Furthermore, UV irradiation of nuclear extracts in vitro did not activate HSF, whereas in vitro heat shock treatment did. These results suggest that HSF activation can be induced by UV irradiation at normal physiological temperature and hsp72 accumulation results from an increased activated HSF level, i.e. a transcriptional

up-regulation of hsp72. In addition, the mechanism responsible for UV-induced HSF activation may differ from the process that operates in heat-treated cells.

L10 ANSWER 34 OF 42 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 25
AU Hang, Haiying; Fox, Michael H.
TI Levels of 70-kDa heat shock protein through the cell cycle in several mammalian cell lines
SO Cytometry (1996), 25(4), 367-373
CODEN: CYTODQ; ISSN: 0196-4763
AB Seven different cell lines were analyzed by flow cytometry to evaluate the variation in heat shock protein hsp70 through the cell cycle. Inducible (hsp72) or both constitutive and inducible (hsp70) heat shock proteins were measured with monoclonal antibodies, and the cell cycle distribution was simultaneously measured with propidium iodide. Cell lines analyzed were Rat-1, HR-24 (Rat-1 transfected with human **hsp72 gene**), CHO (Chinese hamster ovary), C3H-10T1/2, AG1522 (normal human foreskin), GM2149 (normal human female skin), and Hela. None of the cell lines had a substantial variation in hsp72 or hsp70 levels through the cell cycle if they were not heated. In contrast, after chronic heating at 42.0.degree.C for 7.5 h, different cell lines had different patterns of hsp72 or hsp70 through the cell cycle. These results demonstrate that the level of hsp70 is not regulated differentially through the cell cycle in a variety of mammalian cell lines under normal unheated conditions. However, heat shock does induce cell-cycle-specific regulation of hsp70, which varies for different cell lines.

L10 ANSWER 35 OF 42 MEDLINE DUPLICATE 26
AU Murphy S J; Song D; Welsh F A; Wilson D F; Pastuszko A
TI The effect of hypoxia and catecholamines on regional expression of heat-shock protein-72 mRNA in neonatal piglet brain.
SO BRAIN RESEARCH, (1996 Jul 15) 727 (1-2) 145-52.
Journal code: B5L; 0045503. ISSN: 0006-8993.
AB The present study has shown that hypoxia leads to expression of heat-shock protein in the brain of newborn piglets and this process is almost completely abolished by depletion of catecholamines prior to the hypoxic episode. The piglets were anesthetized and mechanically ventilated. One hour of hypoxia was generated by decreasing the oxygen fraction in the inspired gas (FiO2) from 22% to 6%-10%. FiO2 was then returned to the control value for a period of 2 h. Following the 2 h of reoxygenation, regional expression of the 72-kDa heat-shock protein (hsp72) mRNA was determined using in situ hybridization and autoradiography. The hypoxic insult (cortical pO2 = 3-10 mmHg) induced expression of hsp72 mRNA in regions of both white and gray matter, with strong expression occurring in the cerebral cortex of individual animals. Depleting the brain of catecholamines prior to hypoxia, by treating the animals with alpha-methyl-p-tyrosine (AMT), resulted in a major change in the hsp72 mRNA expression. In the catecholamine depleted group of animals, the intensity of hsp72 mRNA expression was greatly decreased or almost completely abolished relative to the nondepleted hypoxic group. These results suggest that the catecholamines play a significant role in the expression of the **hsp72 gene** in response to hypoxic insult in neonatal brain.

L10 ANSWER 36 OF 42 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Lippman, C. (1); Welsh, F.; Kaplitt, M.; O'Connor, W.; During, M.; Mobbs,

C.; O'Connor, M.; Freese, A.
TI AAV-Vector mediated human **HSP72 gene** transfer into cultured cells.
SO Society for Neuroscience Abstracts, (1995) Vol. 21, No. 1-3, pp. 1805. Meeting Info.: 25th Annual Meeting of the Society for Neuroscience San Diego, California, USA November 11-16, 1995
ISSN: 0190-5295.

L10 ANSWER 37 OF 42 MEDLINE DUPLICATE 27
AU Davidson S; Hoj P; Gabriele T; Anderson R L
TI In vivo growth of a murine lymphoma cell line alters regulation of expression of HSP72.
SO MOLECULAR AND CELLULAR BIOLOGY, (1995 Feb) 15 (2) 1071-8.
Journal code: NGY; 8109087. ISSN: 0270-7306.
AB We have identified a murine B-cell lymphoma cell line, CH1, that has a much-diminished capacity to express increased levels of heat shock proteins in response to heat stress in vitro. In particular, these cells cannot synthesize the inducible 72-kDa heat shock protein (HSP72) which is normally expressed at high levels in stressed cells. We show here that CH1 fails to transcribe HSP72 mRNA after heat shock, even though the heat shock transcription factor, HSF, is activated correctly. After heat shock, HSF from CH1 is found in the nucleus and is phosphorylated, trimerized, and capable of binding the heat shock element. We propose that additional signals which CH1 cells are unable to transduce are normally required to activate hsp72 transcription in vitro. Surprisingly, we have found that when the CH1 cells are heated in situ in a mouse, they show normal expression of HSP72 mRNA and protein. Therefore, CH1 cells have a functional **hsp72 gene** which can be transcribed and translated when the cells are in an appropriate environment. A diffusible factor present in ascites fluid is capable of restoring normal HSP72 induction in CH1 cells. We conclude that as-yet-undefined factors are required for regulation of the **hsp72 gene** or, alternatively, that heat shock in vivo causes activation of hsp70 through a novel pathway which the defect in CH1 has exposed and which is distinct from that operating in vitro. This unique system offers an opportunity to study a physiologically relevant pathway of heat shock induction and to biochemically define effectors involved in the mammalian stress response.

L10 ANSWER 38 OF 42 MEDLINE DUPLICATE 28
AU Pannen B H; Maeda K; Ayuse T; Brienza N; Revelly J P; Robotham J L; Buchman T G
TI Hepatic heat shock and acute-phase gene expression are induced simultaneously after celiotomy in the anesthetized pig.
SO ANESTHESIOLOGY, (1995 Oct) 83 (4) 850-9.
Journal code: 4SG; 1300217. ISSN: 0003-3022.
AB BACKGROUND: The liver plays a central role in the whole organism's response to injury. Expression of hepatic acute-phase and heat-shock genes likely contributes to the restoration of homeostasis after stressful events. However, after prolonged ischemia, hepatic transcription of heat-shock genes can exclude the simultaneous transcription of acute-phase genes. The issue of whether hepatic 72-kd heat-shock protein (**hsp72**) **gene** expression is induced under perioperative conditions that do not result in prolonged liver ischemia and whether this might further affect the expression of the acute-phase reactant

inter-alpha-trypsin inhibitor (alpha-Ti) was examined. METHODS: Pigs were anesthetized with sodium pentobarbital and ketamine hydrochloride, tracheally intubated, and their lungs ventilated. After celiotomy, a hepatic biopsy sample was obtained. Arterial blood pressure, cardiac output, and total hepatic blood flow were measured. Subsequent biopsies were obtained at 1, 2, 3, 4, and 6 h after the initial biopsy. Arterial norepinephrine concentrations were measured using high-pressure liquid chromatography. Nuclear runoff (run on) analysis and Northern blotting were applied to estimate changes in hsp72 and alpha-Ti gene transcription rates and RNA levels. Western blotting was used to estimate changes in hsp72 levels. RESULTS: Hemodynamic parameters did not change significantly over time. Arterial norepinephrine concentrations were increased at all time points. Hepatic hsp72 RNA levels increased up to sixfold while nuclear runoff assays did not detect significant changes in **hsp72 gene** transcription rates. The increases in hsp72 RNA levels correlated with accumulation of hsp72 (up to sevenfold). Increases in alpha-Ti transcription rates up to 42-fold were associated with respective increases in alpha-Ti RNA levels (up to 17-fold). CONCLUSIONS: These data demonstrate that hepatic expression of hsp72 is not confined to conditions that lead to prolonged liver ischemia but is also part of the response of the liver to surgery under general anesthesia. Furthermore, these conditions are permissive for the simultaneous RNA expression of the acute-phase reactant alpha-Ti.

L10 ANSWER 39 OF 42 MEDLINE DUPLICATE 29
AU Hayes R L; Yang K; Raghupathi R; McIntosh T K
TI Changes in gene expression following traumatic brain injury in the rat.
SO JOURNAL OF NEUROTRAUMA, (1995 Oct) 12 (5) 779-90. Ref: 59
Journal code: J82; 8811626. ISSN: 0897-7151.
AB This paper reviews changes in gene expression produced by two rodent models of traumatic brain injury: cortical impact injury and fluid-percussion injury. Cortical impact injury produces transient increases in c-fos mRNA expression, which begin as early as 5 min after injury and subsides by 1 day after injury in the cerebral cortex ipsilateral to injury. In addition, AP-1 transcription factor binding is greatly increased in the injured cerebral cortex at 1, 3, and 5 h post-injury. AP-1 binding remains increased for at least 1 day after injury, while SP-1 transcription factor binding activity does not increase. Additional studies have confirmed increases in c-fos mRNA expression in the hippocampus at 30 min, 1 h, and 3 h after injury. These increases in c-fos mRNA in the hippocampus preceded increased levels of NGF mRNA that were detected at 1 and 3 h but not at 30 min following injury. Following fluid-percussion injury, increases in c-fos mRNA can be detected as early as 2 h following injury in the cortex ipsilateral to the site of injury as well as in the hippocampus. Heat-shock protein (hsp72) mRNA is also increased in the ipsilateral cortex and hippocampus following fluid percussion injury. By 24 h post-injury, both c-fos and **hsp72 gene** expression return to control levels. Severe but not moderate fluid percussion injury produces increased gene expression for glucose-regulated proteins (grp78, grp94) 12 h following injury. Fluid-percussion injury also produces significant increases in expression of both interleukin-1 beta and tumor necrosis factor-alpha in the injured cortex and ipsilateral hippocampus as early as 1 h post-injury, that remains elevated up to 6 h in the injured cortex and hippocampus.

L10 ANSWER 41 OF 42 MEDLINE DUPLICATE 31
AU Stege G J; Li L; Kampinga H H; Konings A W; Li G C
TI Importance of the ATP-binding domain and nucleolar localization domain of HSP72 in the protection of nuclear proteins against heat-induced aggregation.
SO EXPERIMENTAL CELL RESEARCH, (1994 Sep) 214 (1) 279-84.
Journal code: EPB; 0373226. ISSN: 0014-4827.
AB Heat treatment of cells results in an increased protein content of nuclei when isolated after the heat treatment (intranuclear protein aggregation).
In a previous study, the role of HSP72 was investigated using Rat-1 fibroblasts stably transfected with the human **HSP72 gene**. It was observed that the expression of human HSP72 in Rat-1 cells (HR cells) confers heat resistance. The initial heat-induced increase in the nuclear protein content was lower in HR cells as compared to the parent Rat-1 cells. In the present communication, the effects of overexpression of intact or mutant human HSP72 in Rat-1 cells on heat-induced increase in intranuclear protein aggregation and their relationship to cells' thermal sensitivity were examined. Four closely related cell lines were used for this study: Rat-1 cells which constitutively expressed the intact human HSP72, or mutant human HSP72 either missing its ATP-binding domain or nucleolar localization domain, and wild type Rat-1 cells. Our results show that expression of the intact human HSP72 or mutant human HSP72 missing its ATP-binding domain confers heat resistance and protects cells against heat-induced intranuclear protein aggregation. On the other hand, cells expressing mutant human HSP72 missing its nucleolar localization domain demonstrated heat shock responses similar to control Rat-1 cells.

L10 ANSWER 40 OF 42 MEDLINE DUPLICATE 30
AU Stege G J; Li G C; Li L; Kampinga H H; Konings A W
TI On the role of hsp72 in heat-induced intranuclear protein aggregation.
SO INTERNATIONAL JOURNAL OF HYPERTHERMIA, (1994 Sep-Oct) 10 (5) 659-74.
Journal code: IJY; 8508395. ISSN: 0265-6736.
AB Heat treatment of cells results in an increased protein content of nuclei and nuclear matrices when isolated after the heat treatment. This increase of TX-100 insoluble protein is interpreted as being the result of protein denaturation and subsequent aggregation. After the heat treatment cells can (partly) recover from these aggregates. Recent data suggest that heat shock proteins (hsps) might be involved in the recovery (disaggregation) from these heat-induced insoluble protein complexes. In this report, the role of hsp72 in the process of aggregation and disaggregation was investigated using: non-tolerant rat-1 cells, thermotolerant rat-1 cells (rat-1 TT), and transfected rat-1 cells constitutively expressing the human inducible **hsp72 gene** (HR-24 cells). After heating the various cells, it was observed that the expression of the human hsp72 confers heat resistance (43-45 degrees C). Heat-induced intranuclear protein aggregation was less in HR and rat-1 TT cells as compared to nontolerant rat-1 cells. After heat treatments leading to the same initial intranuclear protein aggregation, rat-1 TT cells recovered more rapidly from these aggregates, while HR cells recovered at the same rate as nontolerant rat-1 cells. Our data suggest that increased levels of hsp72 can confer heat resistance at the level of initial (nuclear) heat damage. Elevated levels of hsp72 alone, however, do not enable cells to recover more rapidly from heat-induced intranuclear protein aggregates.

L10 ANSWER 41 OF 42 MEDLINE DUPLICATE 31
AU Stege G J; Li L; Kampinga H H; Konings A W; Li G C
TI Importance of the ATP-binding domain and nucleolar localization domain of HSP72 in the protection of nuclear proteins against heat-induced aggregation.
SO EXPERIMENTAL CELL RESEARCH, (1994 Sep) 214 (1) 279-84.
Journal code: EPB; 0373226. ISSN: 0014-4827.
AB Heat treatment of cells results in an increased protein content of nuclei when isolated after the heat treatment (intranuclear protein aggregation). In a previous study, the role of HSP72 was investigated using Rat-1 fibroblasts stably transfected with the human **HSP72 gene**. It was observed that the expression of human HSP72 in Rat-1 cells (HR cells) confers heat resistance. The initial heat-induced increase in the nuclear protein content was lower in HR cells as compared to the parent Rat-1 cells. In the present communication, the effects of overexpression of intact or mutant human HSP72 in Rat-1 cells on heat-induced increase in intranuclear protein aggregation and their relationship to cells' thermal sensitivity were examined. Four closely related cell lines were used for this study: Rat-1 cells which constitutively expressed the intact human HSP72, or mutant human HSP72 either missing its ATP-binding domain or nucleolar localization domain, and wild type Rat-1 cells. Our results show that expression of the intact human HSP72 or mutant human HSP72 missing its ATP-binding domain confers heat resistance and protects cells against heat-induced intranuclear protein aggregation. On the other hand, cells expressing mutant human HSP72 missing its nucleolar localization domain demonstrated heat shock responses similar to control Rat-1 cells.

L10 ANSWER 42 OF 42 MEDLINE DUPLICATE 32

AU Schreiber S S; Najm I; Tocco G; Baudry M

TI Co-expression of HSP72 and c-fos in rat brain following kainic acid treatment.

SO NEUROREPORT, (1993 Dec 13) 5 (3) 269-72.
Journal code: A6M; 9100935. ISSN: 0959-4965.

AB The relationship between heat shock protein 72 (HSP72) and c-fos gene expression following systemic administration of kainic acid was investigated by combining immunocytochemistry for HSP72 with in situ hybridization for c-fos. Increased HSP72 expression was detected in adult rat hippocampus 4 h after seizure-onset. Transient co-expression of c-fos and HSP72 occurred in neurons that are resistant to kainic acid, whereas prolonged co-expression was observed in vulnerable neurons. The spatial distribution and developmental time course of kainic acid-induced HSP72 expression were similar to those of kainic acid-induced neurodegeneration.

The results demonstrate a relationship between c-fos and **HSP72 gene** expression and suggest that prolonged co-expression of these genes plays a role in kainic acid-induced neuronal death.

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     (FILE 'HOME' ENTERED AT 17:14:40 ON 25 JAN 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 17:14:54 ON 25 JAN
     2002
L1          2379 S HSP72
L2       2474033 S DNA OR CDNA OR POLYNUCLEOTIDE
L3           296 S L1 AND L2
L4            92 S (DNA OR CDNA OR POLYNUCLEOTIDE)(8A)HSP72
L5            31 DUP REM L4 (61 DUPLICATES REMOVED)
L6            40 S HSP72(3A)(DNA OR CDNA)
L7            16 DUP REM L6 (24 DUPLICATES REMOVED)

=> d 6-16 au ti so ab l7

L7   ANSWER 6 OF 16     MEDLINE                              DUPLICATE 4
AU   Xia H; Ikata T; Katoh S; Rokutan K; Saito S; Kawai T; Kishi K
TI   Whole body hyperthermia selectively induces heat shock protein 72 in
     neurons of the rat spinal cord.
SO   NEUROSCIENCE LETTERS, (1998 Dec 24) 258 (3) 151-4.
     Journal code: N7N; 7600130. ISSN: 0304-3940.
AB   The aims of this study were to examine whether the heat shock response is
     operative in the spinal cord, and to identify the type of responsible
     cell. Immunoblot analysis using an antibody specific for a highly
     stress-inducible heat shock protein with a molecular mass of 72 kDa
     (HSP72) showed that exposing rats to whole body hyperthermia remarkably
     induced HSP72 protein in the spinal cord within 2 h. Northern blot
     analysis with a cDNA probe for human HSP72
     demonstrated that whole body hyperthermia induced the expression of HSP72
     mRNA within 30 min in the spinal cord. Immunohistochemical analysis
showed
     that neurons in the gray matter appear to be a preferential target of the
     heat shock response, suggesting that the heat shock response might have a
     therapeutic implication for protection against spinal cord injury.

L7   ANSWER 7 OF 16     MEDLINE                              DUPLICATE 5
AU   Kitagawa H; Setoguchi Y; Fukuchi Y; Mitsumoto Y; Koga N; Mori T; Abe K
TI   Induction of DNA fragmentation and HSP72
     immunoreactivity by adenovirus-mediated gene transfer in normal gerbil
     hippocampus and ventricle.
SO   JOURNAL OF NEUROSCIENCE RESEARCH, (1998 Oct 1) 54 (1) 38-45.
     Journal code: KAC; 7600111. ISSN: 0360-4012.
AB   Foreign genes have been successfully transferred and expressed in
     experimental animal brains using adenoviral vectors. However, it is not
     fully understood whether adenovirus-mediated gene transfer causes
     stressful or cytotoxic injury in brain. A replication-defective
adenoviral
     vector containing the Escherichia coli lacZ gene (AdCMVnLacZ) was
directly
     injected into right hippocampus and lateral ventricle of normal gerbil
     brains. Temporal and spatial profiles of the expression of lacZ gene
     products, DNA fragmentation detected by terminal deoxynucleotidyl d-UTP
     nick end labeling (TUNEL) staining, and heat shock protein 72 (HSP72)
     immunoreactivity were examined until 21 days after the injection. In the
     ventricle, lacZ gene was immediately and strongly expressed at 8 hr after
     the injection of AdCMVnLacZ, with a peak at 1-3 days, and disappeared by
     21 days. Although a small number of choroid plexus cells were TUNEL
     positive at 3 and 7 days, no HSP72 immunostaining was observed in the
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ventricle. Small-to-moderate expression of lacZ gene was found in the needle route from 8 hr to 3 days after the injection, and a small number of TUNEL-positive cells were detected at the needle track at 1-3 days. In the hippocampus, lacZ gene was markedly expressed around the dentate gyrus (DG) at 8 hr to 3 days with a peak at 1 day. Large number of TUNEL or moderate-to-dense HSP70 staining cells were also detected in the same area. CA1 neuronal cells just adjacent to the needle route showed TUNEL positivity at 1 to 3 days. However, the TUNEL staining was not associated with lacZ gene expression. The majority of lacZ-expressing cells were discriminated from the TUNEL-positive cells, whereas some were double-positive with HSP72 staining in DG. Cellular loss was observed in the CA1 layer around the needle route. An apoptotic change was morphologically observed in the marginal region of the DG at 1-3 days and in the ventricle at 3-7 days. In the sham control group, TUNEL-positive or HSP72-staining cells were only detected around the needle track including CA1 cells adjacent to the needle route. These data suggest that adenoviral gene transfer may induce direct traumatic injury in the CA1 sector near the needle route, indirect apoptotic cell loss in the DG and ventricle, and stressful effect on the dentate granule cells in association with adenovirus infection in normal gerbil brain.

L7 ANSWER 8 OF 16 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Kato K (Reprint); Yamanaka K; Hasegawa A; Okada S
TI Participation of **Hsp72** in **DNA** damage by dimethylarsenics: Possible relationship between Hsp72 and apoptosis induced in pulmonary cultured cells
SO JAPANESE JOURNAL OF TOXICOLOGY AND ENVIRONMENTAL HEALTH, (FEB 1998) Vol. 44, No. 1, pp. P6-P6.
Publisher: PHARMACEUTICAL SOC JAPAN, 2-12-15-201 SHIBUYA, SHIBUYA-KU, TOKYO 150, JAPAN.
ISSN: 0013-273X.

L7 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2002 ACS
AU Cullen, Katherine E.; Sarge, Kevin D.
TI Characterization of hypothermia-induced cellular stress response in mouse tissues
SO J. Biol. Chem. (1997), 272(3), 1742-1746
CODEN: JBCHA3; ISSN: 0021-9258
AB Cells respond to adverse environmental conditions by expressing heat shock proteins, which serve to protect cells from harmful effects of the stress conditions. In this study, we demonstrated that mice subjected to whole body hypothermia induced the cellular stress response, resulting in the increased expression of hsp72 mRNA in brain, heart, kidney, liver, and lung. We performed a detailed anal. of the major parameters of the stress response and found that cold induction of hsp expression is mediated by heat shock factor 1 (HSF1), which is also responsible for heat induction of the cellular stress response. However, there are differences in the mechanisms of HSF1 activation by hypothermia vs. hyperthermia, as hypothermia does not cause the hyperphosphorylation of HSF1 that is characteristic of heat-activated HSF1.

L7 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2002 ACS
IN Hamel, Josee; Brodeur, Bernard; Martin, Denis; Rioux, Clement
TI Streptococcal heat shock proteins, especially HSP70 and **HSP72**, **cDNA** sequences, antibodies and vaccines, and infection diagnosis,

treatment, and prevention
SO PCT Int. Appl., 155 pp.
CODEN: PIXXD2
AB Novel heat shock proteins (HSPs) of Streptococcus pneumoniae, Streptococcus pyogenes, and Streptococcus agalactiae having apparent mol. masses of 70-72 kDa, immunol. related polypeptides, the nucleotide and derived amino acid sequences of HSP72 of S. pneumoniae, the nucleotide and
derived amino acid sequences of HSP70 of S. pyogenes, the nucleotide and derived amino acid sequences of HSP 70 of S. agalactiae, antibodies that binds to the HSPs, and recombinant DNA methods for the prodn. of the HSPs and immunol. related polypeptides are described. The polypeptides, DNA sequences and antibodies of this invention provide new means for the diagnosis, prevention and/or treatment of Streptococcal disease.

L7 ANSWER 11 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Chen, M. (1); Clark, R. S. B.; Kochanek, P. M.; Chen, J.; Stetler, R. A.; Basta, K.; Marion, D. W.; Dekosky, S. T.; Simon, R. P.; Graham, S. H.
TI Regional pattern of 72 kD heat shock protein (**HSP72**) and in situ **DNA** fragmentation in neurons after severe contusive brain injury in rats.
SO Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 1185.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience Washington, D.C., USA November 16-21, 1996
ISSN: 0190-5295.

L7 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2002 ACS
AU Muramatsu, Tsutomu; Ohno, Haruhiko; Shirai, Toshihiko; Takahashi, Akihisa;
Ohnishi, Takeo
TI DNA-damaging agents induce the 72-kD heat shock protein in SV40 transformed normal human fibroblasts
SO J. Dermatol. (1996), 23(9), 589-593
CODEN: JDMYAG; ISSN: 0385-2407
AB In order to elucidate the involvement of DNA damage in the induction of heat shock proteins (stress proteins), the authors examd. the induction of
72-kD heat shock protein (HSP72) in an SV40-transformed human fibroblast cell line (WI38VA13) which was exposed to various DNA-damaging agents, including 1-(4-amino-2-methyl-5-pyrimidinyl)methyl-3-(2-chloroethyl)-3-nitrosourea hydrochloride, bleomycin hydrochloride, cis-diaminedichloroplatinum(II), mitomycin C, methylmethane sulfonate, N-methyl-N'-nitro-N-nitrosoguanidine, and 4-nitroquinoline N-oxide. Induction of HSP72 was detected by the indirect immunofluorescence method using a monoclonal antibody. All the DNA-damaging agents used in this study induced HSP72 on human fibroblasts. DNA damage is one trigger for the induction of HSP72.

L7 ANSWER 13 OF 16 MEDLINE DUPLICATE 6
AU Manev H; Kharlamov A; Armstrong D M
TI Photochemical brain injury in rats triggers **DNA** fragmentation, p53 and **HSP72**.
SO NEUROREPORT, (1994 Dec 20) 5 (18) 2661-4.
Journal code: A6M; 9100935. ISSN: 0959-4965.
AB The aim of the study was to examine whether apoptosis, apoptosis-related protein p53 and heat-shock protein (HSP) 72 participate in the response of
the brain to focal injury. Male Sprague-Dawley rats received intravenously
a photosensitive dye rose bengal. Unilateral cortical thrombosis was

induced by illuminating the skull of rose bengal-treated rats for 10 min with a focused beam of light. Animals were killed and brains were processed for immunohistochemical detection of **DNA** fragmentation, p53, and **HSP72** kD. **DNA** fragmentation and p53 were increased only in the perifocal area in the cortex ipsilateral to the thrombotic focus, while HSP72 increased throughout the ipsilateral cortex, except in the immediate perifocal area. The results suggest that in response to focal brain injury, some cells die through an apoptotic process that might involve an accumulation of p53.

L7 ANSWER 14 OF 16 MEDLINE DUPLICATE 7
AU Katayama S; Shuntoh H; Matsuyama S; Tanaka C
TI Effect of heat shock on intracellular calcium mobilization in neuroblastoma x glioma hybrid cells.
SO JOURNAL OF NEUROCHEMISTRY, (1994 Jun) 62 (6) 2292-9.
Journal code: JAV; 2985190R. ISSN: 0022-3042.
AB The effect of heat shock on agonist-stimulated intracellular Ca2+ mobilization and the expression of heat shock protein 72 (hsp72) in neuroblastoma x glioma hybrid cells (NG 108-15 cells) were examined. Hsp72 was expressed at 6 h after heat shock (42.5 degrees C, 2 h), reached a maximum at 12 h, and decreased thereafter. Bradykinin-induced [Ca2+]i rise was attenuated to 28% of control by heat shock at 2 h after heat shock, and reversion to the control level was seen 12 h later. When the cells were treated with quercetin or antisense oligodeoxyribonucleotide against **hsp72 cDNA**, the synthesis of **hsp72** was not induced by heat shock, whereas bradykinin-induced [Ca2+]i rise was abolished and the [Ca2+]i rise was not restored. Recovery from this stressed condition was evident when cells were stimulated by the Ca(2+)-ATPase inhibitor thapsigargin, even in the presence of either quercetin or antisense oligodeoxyribonucleotide. Inositol 1,4,5-trisphosphate (IP3) production was not altered by heat shock at 12 h after heat shock, whereas IP3 receptor binding activity was reduced to 45.3%. In the presence of quercetin or antisense oligodeoxyribonucleotide, IP3 receptor binding activity decreased and reached 27.2% of the control 12 h after heat shock. Our working thesis is that heat shock transiently suppresses the IP3-mediated intracellular Ca2+ signal transduction system and that hsp72 is involved in the recovery of bradykinin-induced [Ca2+]i rise.

L7 ANSWER 15 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Ohno, Haruhiko; Takahashi, Akihisa; Yamashina, Yukio; Muramatsu, Tsutomu; Tada, Hideyuki; Kobayashi, Nobuhiko; Yamaji, Masami; Shirai, Toshihiko; Ohnishi, Takeo
TI Induction of **HSP72** by **DNA** damaging agents.
SO Journal of Radiation Research, (1993) Vol. 34, No. 4, pp. 405.
Meeting Info.: 36th Annual Meeting of the Japan Radiation Research Society Hiroshima, Japan October 27-29, 1993
ISSN: 0449-3060.

L7 ANSWER 16 OF 16 MEDLINE DUPLICATE 8
AU Muramatsu T; Tada H; Kobayashi N; Yamaji M; Shirai T; Ohnishi T
TI Induction of the 72-kD heat shock protein in xeroderma pigmentosum complementation group A fibroblasts.
SO JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1992 Nov) 99 (5) 634-8.
Journal code: IHZ; 0426720. ISSN: 0022-202X.

AB In mammalian cells, 72-kD heat shock protein (HSP72) is the major stress-inducible protein that is thought to play a protective role against the various environmental stresses. In order to know the induction mechanism of HSP72, we examined the **HSP72** in **DNA** repair-deficient xeroderma pigmentosum group A fibroblasts (XP2OSSV) and normal fibroblasts (WI38VA13) by the indirect immunofluorescence method using a monoclonal antibody specific for the inducible 72-kD protein. Heat-shock treatment of the same survival fraction (5% survival) induced HSP72 in xeroderma pigmentosum (XP) and normal cells. However, as compared with XP cells, normal cells showed the induction of HSP72 more rapidly and strongly. When XP and normal cells were irradiated with UVC at the same survival dose (10% survival), apparent induction of HSP72 was observed in both cell lines. In the case of UVC irradiation at the same dose (1.0 J/m2), though XP cells showed the induction of HSP72, HSP72 was not induced in normal cells. In both cell lines, heat-shock treatment caused more rapid induction of HSP72 than UV irradiation. These results suggest that the induction mechanism of HSP72 might be different between heat-shock treatment and UV irradiation. In addition, in the case of UV irradiation, the extent of DNA damage after DNA repair or the cell death might be involved in the induction of HSP72.

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